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Postpartum depression and child growth in Tanzania: A cohort study

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Shortened running title: Postpartum depression and child growth: A cohort study.

21 Abstract

22 *Objective:* To examine the association between postpartum depression and child growth in a
23 Tanzanian birth cohort.

24 *Design:* Prospective cohort study.

25 *Setting:* Moshi, Tanzania.

26 *Population:* Pregnant women over the age of 18 who sought antenatal care at two health clinics
27 in Moshi, and the children they were pregnant with, were assessed for inclusion in this study.

28 *Methods:* The women were interviewed twice during pregnancy and three times after birth, the
29 final follow-up taking place 2-3 years postpartum. Signs of postpartum depression were assessed
30 approximately 40 days postpartum with the Edinburgh Postnatal Depression Scale (EPDS).

31 *Main outcome measures:* Child growth was assessed with anthropometric measurements at 2-3
32 years of age, and expressed as mean z-scores.

33 *Results:* 1128 mother-child pairs were followed throughout the duration of the study. 12.2% of
34 the mothers showed signs of postpartum depression. Adjusted mean height-for-age z-score (HAZ)
35 was significantly lower at 2-3 years follow-up for children of mothers with postpartum depression,
36 compared to children of mothers without (difference in HAZ: -0.32, 95%CI:-0.49;-0.15). Adjusted
37 mean weight-for-height z-score (WHZ) was significantly increased for the children exposed to
38 postpartum depression (difference in WHZ: 0.21, 95%CI:0.02;0.40), while there was no significant
39 difference in adjusted weight-for-age z-score (WAZ) (difference in WAZ: -0.04, 95%CI:-0.20;0.12).

40 *Conclusions:* We found that postpartum depressive symptoms predicted decreased linear height
41 in children at 2-3 years of age, and slightly increased weight-for-height.

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43 (29190909), and the Lundbeck foundation (F-61171-19-27).

Keywords: Postnatal depression, maternal depression, child development, nutritional status.

Tweetable abstract: Postpartum depression in Tanzanian mothers is associated with impaired child growth at 2-3 years of age.

Introduction

Postpartum depression (PPD) is a common and debilitating condition among mothers worldwide.

Studies in sub-Saharan Africa have shown incidence of PPD to be 13-35%^(1, 2). Estimates do however vary greatly depending on country and cultural context, and generally seem to be increasing⁽³⁾. According to International Classification of Diseases (ICD-10) PPD is a depression commencing within six weeks of delivery⁽⁴⁾, and involves symptoms such as depressed mood, lack of interest in surroundings, lack of energy, insomnia and low self-esteem⁽⁵⁾.

While PPD clearly has an influence on maternal health, it is also recognized that child mental and physical development could be detrimentally affected by maternal PPD⁽³⁾. Maternal depression is connected to early interaction problems between mother and child and inadequate caregiving, such as non-exclusive or early cessation of breastfeeding, decreased preventative healthcare seeking behaviour and undesirable sleep patterns^(1, 3, 5-7). Children of depressed mothers are more likely to get diarrhoea and other childhood infections, and are exposed to more stressors such as family conflicts, both of which have been shown to influence child growth⁽⁵⁻⁷⁾.

These shortcomings in parental care ability are particularly likely to impact physical child health in low income countries (LICs), due to harsher conditions, including food insecurity and sanitation issues; especially during the postpartum period, where the infant is completely dependent on their main caregiver, often the mother⁽⁵⁾.

Considering these possible pathways, it seems reasonable that depressive symptoms in the postpartum period could impair physical child health and growth, particularly in LICs. Previous research generally supports this hypothesis, but there is a call for more longitudinal studies in order to investigate causality and pathways^(1, 5, 8). Most of the current research for LICs is from Asia, and there is a definite information gap for sub-Saharan Africa. The aim of this study is to examine the association between PPD and child growth in a Tanzanian birth cohort.

Methods

This prospective cohort study took place in Moshi, in the Kilimanjaro region of northern Tanzania, and ran from the 1st of March 2014 to 30th of June 2017.

Population

Pregnant women attending antenatal care at either of two primary care facilities were assessed for eligibility. The primary care facilities Majengo and Pasua were chosen, due to their high attendance rate of pregnant women.

All consenting pregnant women who sought antenatal care in the aforementioned facilities during the enrolment period, who were aged 18 or above at the time, and whose gestational age was below 30 weeks, were eligible for inclusion. Women with multiple or molar pregnancies, or who planned to give birth outside of Moshi, were excluded. Likewise, mother-child pairs were excluded if either party died prior to conclusion of the study, or if core information, such as the child's sex or age, or mother's exposure to PPD, was missing from the data set. Comprehensive contact information was gathered for the participants, and they were reminded of their appointments beforehand, by phone call and home visit, to minimize loss to follow-up. All participants received

compensation for their travel expenses. Participants who moved away during the study were encouraged to come back for follow-up appointments, e.g. when visiting Moshi for family events.

Measures

Included mothers were interviewed five times during the study. The first interview took place at enrolment, and the next at 34 weeks gestational age, then 48 hours post-delivery, 40 days post-delivery, and 2-3 years post-delivery. Gestational age was determined with ultrasound examination at enrolment by a trained nurse. Sociodemographic and reproductive health information was collected at enrolment. Details on exposure to intimate partner violence (IPV) and HIV status were collected at the second interview. The questions relating to IPV were adopted directly from the Swahili version of the “WHO Multi-Country Study on Women and Domestic Violence against Women” as used in the Tanzania Demographic and Health Survey 2010⁽⁹⁾. Birth outcomes were collected in the third interview, and information on breastfeeding practices in the fifth. Interviews were conducted by six trained research nurses, who were native Swahili speakers. The nurses endeavoured to interview the same mothers throughout the study, to create a consistent and safe environment for them, and were specifically trained in handling sensitive subjects, such as emotional health and IPV. A small number of participants were involved in pilot-testing of questionnaires. No CROWN initiative core outcome sets (COS) were used in this study, as no relevant COS currently exist or are under development.

Depression

Information about signs of PPD was collected during the fourth interview, at 40 days postpartum. A locally translated and pilot-tested Swahili version of the Edinburgh Postnatal Depression Scale (EPDS)^(10, 11) was used, as the EPDS is the most commonly used screening tool for postpartum depression, in both high- and low-income countries^(12, 13), and because it is reliable and cost-

effective⁽³⁾. The EPDS was translated from English to Swahili by a native speaker. Ten individual interviews were conducted to assess the quality of the translation and determine whether local women found the questions meaningful. Based on feedback from the interviewed women, a revised translation was developed and tested in another round of ten interviews, before being accepted as the final version used in this study. The EPDS consists of 10 questions, each of which addresses a clinical symptom of depression. Each question can score from 0-3 with three representing the most severe option, leading to a maximum score of 30. Based on previous literature, a cut-off value of 13 was selected to indicate probable postpartum depression⁽¹²⁾. Signs of maternal depression were also collected during pregnancy at 34 weeks gestational age to assess signs of antenatal depression, and at the final interview 2-3 years after delivery to assess signs of depression at 2-3 years follow-up, using the same methodology as described above.

Growth

Child growth indicators were assessed in conjunction to the final interview, where anthropometric measurements were taken of both mother and child. Child growth was expressed through mean height-for-age z-scores (HAZ), weight-for-age z-scores (WAZ) and weight-for-height z-scores (WHZ)⁽¹⁴⁾. Z-scores describe how many standard deviations an individual or population is from a standard population mean. When z-scores are calculated, we take into account height, weight, age, and sex, meaning that z-scores can be interpreted independently of sex and age⁽¹⁴⁾. The age of each child was calculated at the final follow-up interview, as time between date of birth and date of final interview, and height and weight were measured during the same visit. Following WHO guidelines, children of less than 24 months age had their length measured lying down, and children ages 24 months or older had their height measured standing⁽¹⁴⁾. A measuring board was used for the supine measurements, while a stadiometer was used for the standing measurements.

Weight measurements were performed with an electronic weighing scale. All measurements were made by trained research nurses. Information on child sex was collected in the 48 hours post-delivery interview. Calculations of z-values were done with the WHO Anthro computer program, version 3.2.2, using the WHO standard population as the reference population⁽¹⁵⁾.

Statistical analysis

All data was double-entered. Statistical analyses were performed with the Stata software package (version 15). To determine whether there were any major unexpected differences between the exposed and unexposed groups, Pearson chi-square and Fischer's exact test were used as appropriate, to compare cohort characteristics.

To analyse the association between signs of maternal PPD and each of the three child growth indicators, linear regression was performed. A theoretical framework, i.e. a directed acyclic graph (DAG)⁽¹⁶⁾, was developed to assess the potential causal and confounding pathways involved in this study (figure S1). The DAG network was developed with the DAGitty web-based computer program, version 2.3⁽¹⁷⁾, and was used a priori to determine which variables to adjust for in the final regression model. This was done by applying an algorithm within the DAGitty program which determines the optimal set of potential confounders to adjust for in order to minimize bias in the regression. The algorithm seeks to identify the smallest possible set of variables that collectively block all biasing paths in the network. In our network this set consisted of maternal HIV status, child HIV status, maternal age and maternal exposure to IPV during pregnancy, wherefore these confounders were chosen to be adjusted for in the final regression model. For practical reasons, a priori chosen potential confounding variables were removed from the model if the number of observations was so low that the statistical power suffered, and there was no obvious difference in observations of the potential confounding variable between the groups.

The confounders included in the final regression model were assessed for signs of effect modification. If they showed any such signs, they were included as variables in a stratified analysis, so the amount and direction of the effect modification could be ascertained. In an effort to assess how depression at other time points affected the relationship between PPD and child growth, antenatal depression and depression at 2-3 years follow-up were included in the stratified analysis. The selection of variables took place a priori.

Ethical considerations

Ethical approval was obtained and all included mothers gave informed consent. The WHO ethical and safety recommendations for research on domestic violence against women were followed⁽¹⁸⁾. If the mothers reported IPV, they were offered referrals to relevant institutions. If they showed signs of PPD, they were offered counselling at local health centres. Severe cases were referred to a regional hospital where further care, including antidepressant medication (Amitriptyline), was available. The use of antidepressants is not common in Tanzania and to our knowledge none of the mothers in this study received medication for their PPD.

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Results

Figure 1 shows the participant inclusion flowchart. Of the women who attended antenatal care at the study sites, 1300 were deemed eligible for inclusion and were willing to participate. During the study period 129 mother-child pairs were excluded as they no longer fulfilled the inclusion criteria, leaving a true cohort size of 1171 mother-child pairs. The cohort had a follow-up rate of 96.3%.

The descriptive cohort characteristics for the exposed and the unexposed mothers and children are shown in table 1. Of the 1128 interviewed mother-child pairs, 12.2% were exposed to maternal signs of PPD, while 87.8% were not. All mothers reported being either married or in a relationship at enrolment.

Table 2 shows the crude and adjusted differences in mean for the three growth outcomes HAZ, WAZ, and WHZ. For the crude regression, only HAZ shows a statistically significant difference between the PPD exposed and non-exposed groups, with the children in the exposed group being significantly shorter for their age than the children in the unexposed group (-0.27, 95%CI:-0.43;-0.10). The adjusted regression was controlled for the a priori defined potential confounders maternal age, maternal HIV status and maternal exposure to IPV during pregnancy. As only 0.4% of the children were HIV positive, child HIV status was left out of the model due to lack of power. In the adjusted model the difference remained highly significant for HAZ (-0.32, 95%CI:-0.49;-0.15), while WHZ achieved marginal statistical significance, indicating that the children in the exposed group were heavier for their height than the children in the unexposed group (0.21, 95%CI:0.02;0.40).

The results of the stratified analyses are presented in table 3. Of the confounders included in the final regression model, maternal age and exposure to IPV both appeared to be effect modifiers and were therefore included in the stratified analysis along with antenatal depression and depression at 2-3 years follow-up. The table shows stratified results for HAZ, WAZ and WHZ, with maternal exposure to PPD being the main independent variable of interest.

The association between PPD and HAZ retained the same directionality in all subgroups, but to dissimilar extents. Three of the four stratification variables showed clear effect modification. The association between PPD and HAZ was stronger among mother-child pairs where mothers were

exposed to IPV during pregnancy (-0.50, 95%CI:-0.75;-0.25), than among those without IPV exposure. Similarly, the association was stronger among mothers over the age of 30 (-0.49, 95%CI:-0.81;-0.16) and among those who had depression at 2-3 years follow-up (-0.87, 95%CI:-1.54;-0.20). The fourth stratification variable also suggested effect modification, but not as clearly. The association between PPD and HAZ was not dramatically different between those with antenatal depression and those without, yet the statistical significance was only retained in the group with no antenatal depression (-0.34, 95%CI:-0.54;-0.14). With regard to PPD and WAZ, only maternal age showed any signs of effect modification, with significantly decreased WAZ in the oldest subgroup (-0.39, 95%CI:-0.73;-0.05). There were signs of effect modification for all variables when considering the association between PPD and WHZ. The association was stronger in the subgroups exposed to IPV (0.37, 95%CI:0.10;0.63) and antenatal depression (0.44, 95%CI:0.05;0.84). Depression at 2-3 years follow-up also trended towards a stronger association between PPD and WHZ for the exposed subgroup, but the association was only significant in the unexposed subgroup (0.20, 95%CI:0.01;0.40). Stratifying by maternal age showed a substantial difference in the PPD and WHZ association, where the children of the younger mothers with PPD were heavier for their height than those of younger mothers without PPD, while the children of older mothers with PPD were lighter for their height than their peers. The association however only remained significant for the mothers aged 20-30 years (0.33, 95%CI:0.10;0.57).

Discussion

Main findings

This prospective cohort study shows that maternal exposure to PPD is associated with poorer child growth outcomes at 2-3 years of age. The children of exposed mothers were found to be significantly shorter for their age and heavier for their height, compared with their unexposed peers. The stratified analyses suggested that these relationships were particularly strong among mothers exposed to IPV during pregnancy, mothers over the age of 30, and mothers with depressive symptoms at 2-3 years follow-up. The relationship between PPD and HAZ seemed to be slightly weaker among mothers with signs of antenatal depression.

Somewhat surprisingly, there was no significant difference in exclusive breastfeeding rate between the PPD and non-PPD groups. This implies that breastfeeding did not play a part in creating the growth difference in this population.

Strengths and limitations

This study is strengthened by its prospective cohort design. The study population was larger than any previous cohort studies concerning PPD and child growth in sub-Saharan Africa^(2, 7), and the follow-up rate was >96% after three years. The high follow-up rate was achieved partly by diligently reminding participants to return for their follow-ups, and partly because the mothers were appreciative of the services they received in the study. The study population is representative of the Kilimanjaro region of Tanzania, as can be seen when comparing the cohort to region specific indicators, such as HAZ -1.1, WAZ -0.4 and WHZ 0.3⁽¹⁹⁾, which correspond to the mean growth indicators in this study.

The study used the DAG method for confounder choice, which is a method that has gained momentum within the past ten years. It strengthens the study, as it is a mathematically robust

way of minimizing bias^(16, 17). It is easily reproduced and avoids many problems associated with traditional methods of bias reduction^(16, 17). The main limitation is that the DAG is a simplified representation of reality⁽¹⁶⁾. While our DAG has been thoroughly researched, it cannot be said to show the one true causal network. It will change as our knowledge grows and with differing levels of intricacy included in the network. While we believe our DAG is adequate and that the merits of this method outweigh the limitations, we acknowledge that the choice of confounders is only as strong as the DAG.

The use of EPDS to measure maternal depression could be construed as a minor limitation of this study, as it is a screening tool and cannot diagnose PPD. However, conclusions obtained from other studies are similar to each other, regardless of whether EPDS or diagnostic interviews are used. This, combined with the availability and affordability of the EPDS, speaks strongly for its use as proxy measure for PPD⁽¹³⁾. Another limitation is the possible under-reporting of PPD and IPV, as there is cultural stigma associated with both subjects. We have addressed this problem by training our interviewers in handling sensitive subjects and conducting interviews privately. If under-reporting took place despite our efforts, this would likely lead to an underestimation of the association.

Interpretation

The findings in this study are consistent with those of previous research, especially for LICs. A systematic review of 20 longitudinal studies from low and high income countries found that children of depressed mothers were more likely to be stunted (HAZ <-2) or underweight (WAZ <-2) in their first year of life, but that only HAZ continued to be impaired beyond the first year⁽¹³⁾. This supports our finding that HAZ is affected at 2-3 years of age, while WAZ is not. This makes sense,

270 as WAZ can be improved at any point with appropriate nutrition, whereas low HAZ reflects the
271 cumulative effects of chronic malnutrition.

272 Similarly, a meta-analysis of 17 studies in LICs found that children of mothers with depressive
273 symptoms were more likely to be underweight or stunted than their peers⁽⁸⁾. The studies varied in
274 design and measured exposure and outcome at different times. This mix of timings could explain
275 why both HAZ and WAZ are significantly affected in the meta-analysis, while our follow-up at 2-3
276 years of age only reflects the situation beyond the first year of life.

277 So far only six other studies have been conducted on the subject of PPD and child growth in sub-
278 Saharan Africa^(1,2,6,7,20,21). Collectively, they indicate that maternal PPD negatively impacts HAZ, but
279 probably not WAZ or WHZ. Thus they support our finding that HAZ was significantly lower in the
280 exposed group, as well as the null finding for WAZ. Only one of the studies showed results for
281 WHZ, and these were, in contrast to our study, null⁽⁷⁾. Our study shows a slightly increased WHZ
282 for children of mothers with PPD, but as HAZ, WAZ and WHZ are closely connected, a negatively
283 different height-for-age and a non-different weight-for-age, must give a positively different
284 weight-for-height, and this result is therefore not surprising.

285 The stratified analyses suggest that the association between PPD and child growth is stronger in
286 some subgroups than in others. The large number of secondary analyses begets a risk of
287 spuriousness, which must be considered when interpreting the stratified results. Little evidence
288 exists on the effect modifiers investigated in this paper, but the previously mentioned meta-
289 analysis did find that the worse the depression, the more severe the growth deficit⁽⁸⁾. This
290 supports our finding that while children of mothers with signs of PPD generally have lower HAZ
291 than their peers, the deficit is larger for those with signs of depression both postpartum and at 2-3
292 years follow-up. Having depression at both time points could indicate more chronic depression, or

frequent relapses. Another aspect in support of our finding is that it reached statistical significance despite the small subgroup size (only 2.4% of the mothers showed signs of depression at 2-3 years follow-up).

The limited evidence available points towards the association between PPD and child growth being independent of antenatal depression⁽¹³⁾. The association did not differ much between the antenatal depression subgroups, which indicates independence. However, the association only reached statistical significance in the subgroup without signs of antenatal depression, which in turn could imply that the association is with perinatal depression, rather than PPD alone, or it could simply be due to a loss of statistical power (only 10.2% of the mothers showed signs of antenatal depression).

No supporting literature was found for IPV exposure and maternal age. While our data suggests that the association between PPD and poor child growth is stonger among mothers exposed to IPV during pregnancy and older mothers, these results need verification from further studies.

Conclusion

This study contributes to the body of evidence indicating that increased focus on maternal mental health will benefit both maternal and child health. We found that postpartum depressive symptoms predicted decreased linear height in children at 2-3 years of age, and slightly increased weight-for-height. This suggests that some growth deficits could be avoided by treating maternal depressive symptoms. We propose that future research explores this possibility by performing PPD treatment intervention studies to see if child growth deficits can be avoided, focusing on culturally appropriate and affordable treatments, to increase their applicability in LICs.

We suggest that screening for PPD be included in health policies, and that primary care providers monitor growth more closely for children whose mothers show signs of PPD.

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Disclosure of interests

No competing interests to disclose. Completed disclosure of interest forms are available to view online as supporting information.

Contribution to authorship

Conception: TG, DWM, RM, DM, VR

Planning: TG, DWM, RM, DM, VR, GNS, JJR, CEH, FKM

Data collection: CEH, FKM, GNS, JJR

Analysis: CEH, FKM, VR

Writing manuscript: CEH

Supervision: VR, GNS

336 Details of ethical approval

337 Ethical approval was granted from the Institutional Review Board of the Kilimanjaro Christian
338 Medical University College of the Tumaini University, Makumira, Tanzania (permit no. 592) on
339 January 17th 2014 with extensions granted on March 19th 2015 and January 10th 2017.

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Table/figure caption list

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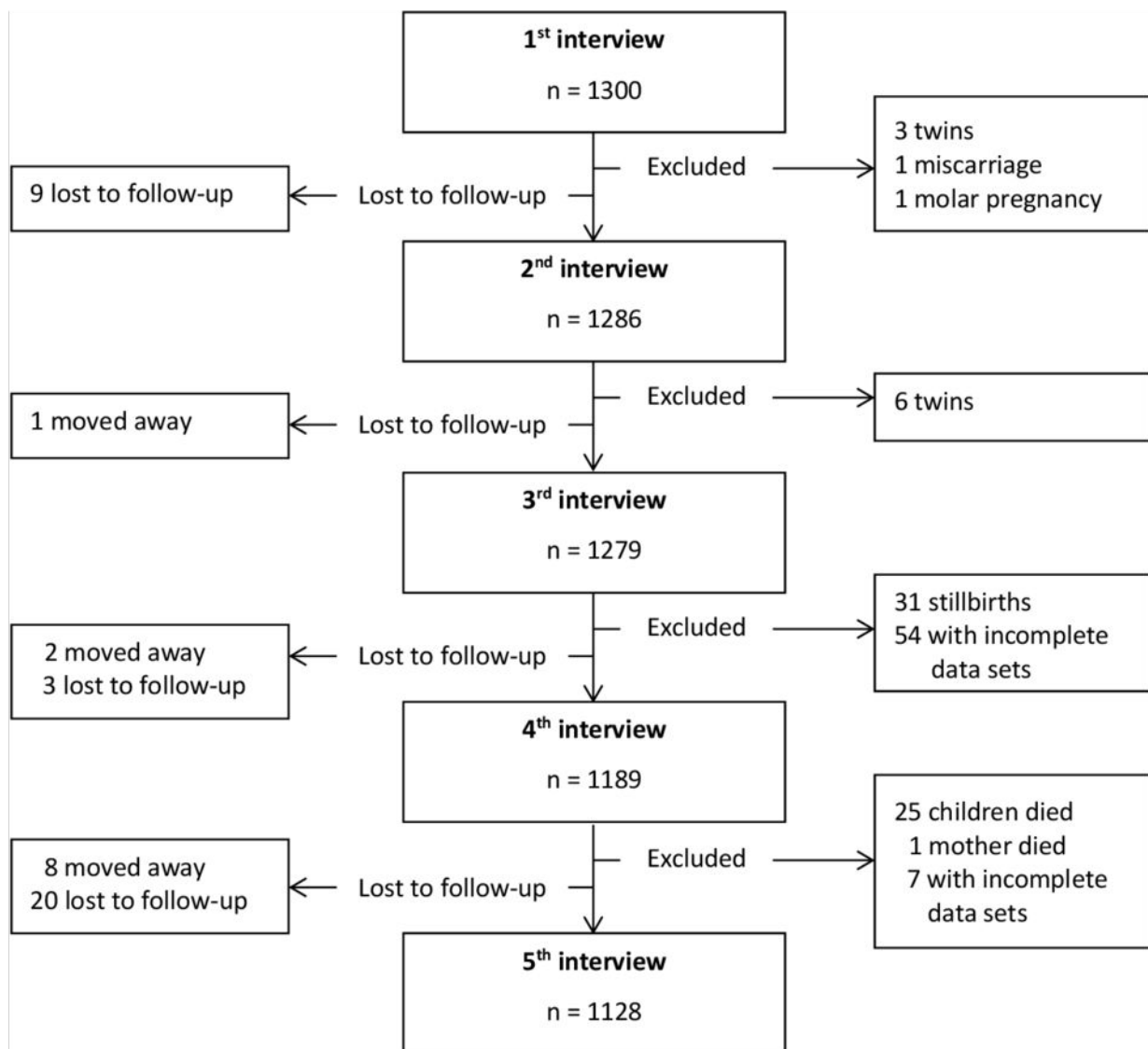


Figure 1. Participant inclusion flowchart. For the 1st and 2nd interview, n represents number of pregnant women. For the 3rd, 4th and 5th interview, n represents the number of mother-child pairs.

Table 1 - Summary of cohort characteristics.

Characteristics	No. of mother-offspring pairs (% of those with characteristic)						P-value
	Total - 1128 pairs		No signs of PPD (EPDS <13) - 990 pairs		Signs of PPD (EPDS ≥13) - 138 pairs		
Maternal							
Age at enrolment †							
<20 years	137	(12.1)	125	(12.6)	12	(8.7)	0.053
20-30 years	740	(65.6)	655	(66.2)	85	(61.6)	
>30 years	251	(22.3)	210	(21.2)	41	(29.7)	
Level of education at enrolment ‡							
Less than primary	24	(2.1)	20	(2.0)	4	(2.9)	0.041
Primary	725	(64.3)	623	(62.9)	102	(73.9)	
Secondary	323	(28.6)	295	(29.8)	28	(20.3)	
Above secondary	56	(5.0)	52	(5.3)	4	(2.9)	
Smoking ‡							
Any smoking during pregnancy	4	(0.4)	2	(0.2)	2	(1.4)	0.076
Alcohol †							
Any alcohol intake during pregnancy	128	(11.3)	107	(10.8)	21	(15.2)	0.126
Height at 2-3 years follow-up ‡							
<150 cm	62	(5.5)	52	(5.3)	10	(7.2)	0.422
150-170 cm	1041	(92.3)	917	(92.6)	124	(89.9)	
≥170 cm	25	(2.2)	21	(2.1)	4	(2.9)	
Weight at 2-3 years follow-up †							
<55 kg	399	(35.4)	352	(35.6)	47	(34.1)	0.741
55-65 kg	345	(30.6)	305	(30.8)	40	(29.0)	
≥65 kg	384	(34.0)	333	(33.6)	51	(37.0)	
Parity at baseline †							
0	459	(40.7)	420	(42.4)	39	(28.3)	0.003
1	335	(29.7)	292	(29.5)	43	(31.2)	
2	213	(18.9)	174	(17.6)	39	(28.3)	
≥3	121	(10.7)	104	(10.5)	17	(12.3)	
HIV status †							
Positive	46	(4.1)	34	(3.4)	12	(8.7)	0.003
Exposure to IPV during pregnancy †							
Reported any IPV	333	(29.5)	262	(26.5)	71	(51.4)	< .001
Signs of antenatal depression †							
(EPDS ≥13)	115	(10.2)	73	(7.4)	42	(30.4)	< .001
Signs of depression at 2-3 years follow-up †							
(EPDS ≥13)	27	(2.4)	19	(1.9)	8	(5.8)	0.005
Child							
Sex †							
Female	549	(48.7)	484	(48.9)	65	(47.1)	0.694
Birth weight †							
<2500g	65	(5.8)	56	(5.7)	9	(6.5)	0.912
2500-4000g	1011	(89.6)	888	(89.7)	123	(89.1)	
≥4000g	52	(4.6)	46	(4.6)	6	(4.3)	
Preterm birth †							
<37 weeks	65	(5.8)	53	(5.4)	12	(8.7)	0.114
Child age at 2-3 years follow up ‡							
<24 months	6	(0.5)	6	(0.6)	0	(0.0)	< .001
24-30 months	658	(58.3)	603	(60.9)	55	(39.9)	
≥30 months	464	(41.1)	381	(38.5)	83	(60.1)	
Confirmed HIV status at 2-3 years follow-up ‡							
Positive	5	(0.4)	4	(0.4)	1	(0.7)	0.497
Exclusive breastfeeding †							
≥ 6 months	551	(48.8)	490	(49.5)	61	(44.2)	0.244

PPD: Postpartum depression, EPDS: Edinburgh postnatal depression scale, BMI: Body mass index, HIV: Human immunodeficiency virus, IPV: Intimate partner violence.

† Difference between groups tested with Pearson's Chi-squared test.

‡ Difference between groups tested with 2-sided Fisher's exact test.

Table 2 - Crude and adjusted difference in mean z-scores and 95% CI by exposure to postpartum depression.

	Crude analysis			Adjusted analysis †	
	Mean z-score (SD)	Difference in mean z-score	95% CI	Difference in mean z-score	95% CI
Height for age z-score (HAZ)					
<i>No signs of postpartum depression</i>	-0.94 (0.94)				
<i>Signs of postpartum depression</i>	-1.21 (0.89)	-0.27	(-0.43; -0.10)	-0.32	(-0.49; -0.15)
Weight for age z-score (WAZ)					
<i>No signs of postpartum depression</i>	-0.34 (0.87)				
<i>Signs of postpartum depression</i>	-0.38 (0.86)	-0.04	(-0.19; 0.12)	-0.04	(-0.20; 0.12)
Weight for height z-score (WHZ)					
<i>No signs of postpartum depression</i>	0.19 (1.04)				
<i>Signs of postpartum depression</i>	0.37 (0.93)	0.17	(-0.01; 0.35)	0.21	(0.02; 0.40)

SD: Standard deviation, CI: Confidence interval.

† Adjusted for maternal age, maternal HIV status and maternal exposure to intimate partner violence during pregnancy.

Table 3 - Stratified analysis of maternal exposure to postpartum depression and its association to child z-scores.

Indicator	Height for age z-score		Weight for age z-score		Weight for height z-score	
	Difference in mean ‡	95%CI	Difference in mean ‡	95%CI	Difference in mean ‡	95%CI
IPV during pregnancy §						
No	-0.15	(-0.39 ; 0.08)	-0.05	(-0.27 ; 0.18)	0.07	(-0.19 ; 0.34)
Yes	-0.50	(-0.75 ; -0.25)	-0.03	(-0.25 ; 0.18)	0.37	(0.10 ; 0.63)
Maternal age ¶¶						
<20 years	-0.20	(-0.75 ; 0.35)	0.20	(-0.26 ; 0.67)	0.47	(-0.12 ; 1.05)
20-30 years	-0.27	(-0.48 ; -0.05)	0.07	(-0.12 ; 0.27)	0.33	(0.10 ; 0.57)
>30 years	-0.49	(-0.81 ; -0.16)	-0.39	(-0.73 ; -0.05)	-0.15	(-0.53 ; 0.23)
Antenatal depression ¶¶¶						
No	-0.34	(-0.54 ; -0.14)	-0.10	(-0.29 ; 0.08)	0.14	(-0.08 ; 0.36)
Yes	-0.27	(-0.62 ; 0.09)	0.15	(-0.17 ; 0.48)	0.44	(0.05 ; 0.84)
Depression at 2-3 years follow-up ¶¶¶						
No	-0.30	(-0.48 ; -0.13)	-0.04	(-0.20 ; 0.12)	0.20	(0.01 ; 0.40)
Yes	-0.87	(-1.54 ; -0.20)	-0.18	(-1.07 ; 0.71)	0.42	(-0.57 ; 1.40)

CI: Confidence interval. IPV: Intimate partner violence.

‡ Difference in mean z-score between the group exposed to postpartum depression and the unexposed group, stratified by five indicators of interest.

§ Stratified analyses adjusted for maternal age and maternal HIV status.

¶¶ Stratified analyses adjusted for maternal HIV status and maternal exposure to intimate partner violence during pregnancy.

¶¶¶ Stratified analyses adjusted for maternal age, maternal HIV status and maternal exposure to intimate partner violence during pregnancy.